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ORIGINAL ARTICLES.

CLINICAL OBSERVATIONS ON BLOCK OF THE BRANCHES OF THE AURICULOVENTRICULAR BUNDLE.

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This report is based on clinical observations that were made on thirty-five patients in each of whom the probable diagnosis of bundle branch block was made from the electrocardiogram. The *QRS* group was atypical in each of the three leads and exceeded 0.1 second in duration. Some of these patients came under observation during the time that studies[1 2] were being made on experimental lesions of the branches of the auriculoventricular bundle of the dog, so that in this clinical study it was natural to attempt to correlate as nearly as possible the electrocardiographic findings with those produced experimentally in the dog.

Age Incidence. Twelve patients were in the fifth and 13 in the sixth decade of life; 6 were under fifty and 4 above seventy; 20 were between fifty-five and sixty-five years. The youngest patient was twenty-three years old.

Possible Etiological Factors. In 17 there was an associated chronic nephritis. Five had arterial hypertension with negative urinary findings. The blood Wassermann test was made in 10. In five of these the reaction was positive. Each of these gave a history of having had a chancre. It was noted that the age incidence in these 5 was below fifty years. Ten gave histories of having had inflammatory rheumatism. Five had been addicted to alcohol and 2 had had typhoid fever.

Symptoms. Eighteen were short of breath on slight exertion. Seventeen had difficult breathing even when at rest. In 8 the duration of the shortness of breath had existed for more than two years, in 10 more than one year and in the remaining 17 less than one year. Twelve had cardiac asthma. Ten gave histories of having anginal pain. In 4 of these the pain had at one time been very severe, lasting several hours, and morphin was required for relief. Twenty had noticed swelling of the feet.

Physical Findings. In every instance the cardiac dulness was increased to the left. Twenty-five had an apical systolic murmur. Ten had a systolic murmur over the aortic area. Only one had the diastolic murmur of aortic regurgitation. This patient had a distinct widening of the aorta. Twelve had gallop rhythm. A muffled first tone was a common finding. Twenty had rales in the bases of the lungs, a palpable liver and some edema of the ankles. Twenty-three had a systolic blood-pressure ranging from 175 to 250. In eight of these a *pulsus alternans* was noted. Seventeen had albumin and granular and hyaline casts in the urine.

Clinical Course Since First Seen by Us. We have been able to follow 21 of these patients. Twelve died within eighteen months after the first examination. The average duration of life was less than one year. Ten died from cardiac failure. One of these died suddenly following recovery from a prostatectomy, presumably a coronary accident. Another after a cataract operation died from a thrombosis of the cerebral arteries. The youngest patient of the series died on the same day after the resection of the left fifth rib for an adhesive pericarditis. The clinical diagnosis of extensive cardiac hypertrophy, adhesive pericarditis, double mitral lesion, tricuspid regurgitation, fibrous myocarditis and cirrhosis of the liver was verified by necropsy. Autopsies were done in 2 other cases in which an extensive sclerosis of the coronary arteries and a disseminated myocardial fibrosis were found. In neither case was the conduction system examined histologically. Six of the 10 patients that are known to be alive are in bad condition. Two of them have had major operations. Following the operations they had very stormy periods of cardiac upset. Since recovery the clinical manifestations of cardiac weakness have rapidly progressed. Four patients have reported that they were improved.

Electrocardiographic Findings. The prevailing ventricular phases were broad at the base and notched or slurred near or at the apex. The duration of the *QRS* group in all instances exceeded 0.1 second and in many the duration approached 0.15 second. The *T*-wave assumed a direction opposite to that of the most prominent ventricular deflection. In thirty-two instances the chief ventricular wave (Fig. 1, top) was positive in lead I and negative in lead III.

In the remaining three the *S*-wave was the most prominent wave in lead I and the *R*-wave in lead III (Fig. 1, bottom).

The fiber of the galvanometer was standardized so that 3 millvts. caused a deflection of 30 mm. In 12 the amplitude of both the *R* and the *S* deflections was large and in 10 small. In 3 the amplitude of the low deflection curves increased while the patients were under observation. One of these patients has been studied over a period of one year. During this time electrocardiograms have been taken at frequent intervals. When first seen he was orthopneic, had

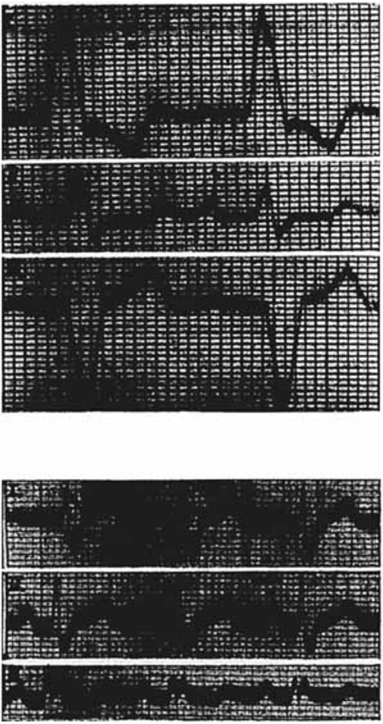

Fig. 1.—*Top:* The prevailing ventricular phase is positive in leads I and II and negative in III; it is blunt or slurred at the apex, and its duration is about 0.14 second. *Bottom:* The chief ventricular deflection is negative in lead I and positive in lead III. The duration of the *QRS* group is about 0.12 second.

passive congestion of the lungs, engorgement of the liver and edema of the ankles. He had a systolic murmur at the apex and a gallop rhythm. The systolic blood-pressure was 238 and the diastolic 120. There was a *pulsus alternans*. He was kept in the hospital until the heart improved and afterward rested at home four months. The initial electrocardiogram (Fig. 2) shows very atypical broad *R* and *S* deflections of low amplitude. In the later electrocardiograms (Figs. 2, 3, 4) the *R* and *S* waves become less atypical and their amplitude gradually increases. In the last record (Fig. 4) the prevailing ventricular deflection is tall and resembles that of a left ventricular preponderance. The duration of the *QRS* group is, however, still prolonged. This change in the electrocardiogram was coincident with the clinical improvement of the patient. He was relatively free from cardiac symptoms for three months, and during that time worked as a

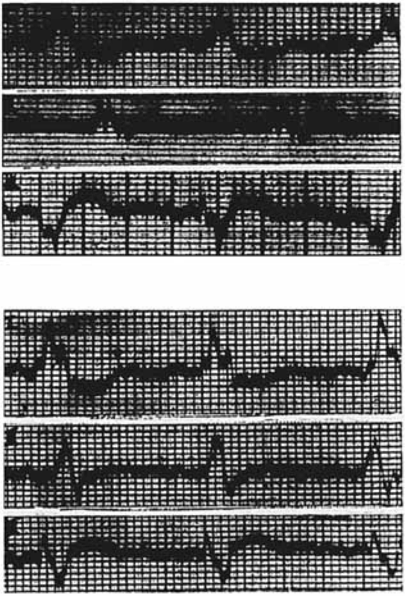

Fig. 2.—*Top:* Initial electrocardiogram taken December 20, 1919. *Bottom:* This electrocardiogram was taken January 5, 1920. The patient had been at rest in bed for two weeks.

presser. Later he became short of breath again and the amplitude of the ventricular deflections decreased. He is at present in the hospital in a critical condition. The electrocardiogram now is similar to that when first seen.

The electrocardiogram (Fig. 5) in one case showed some *QRS* groups that approached the normal in duration. We were able to follow this case, and the *R*-waves of leads I and II were later apparently normal in duration, while the *S*-wave in lead III was slightly broad (Fig. 5, bottom). When first seen at the cardiac clinic of the Central Free Dispensary this patient was in an extremely bad condition. She was cyanotic and very dyspneic. The heart-rate was about 120 per minute and the auricles were fibrillating.

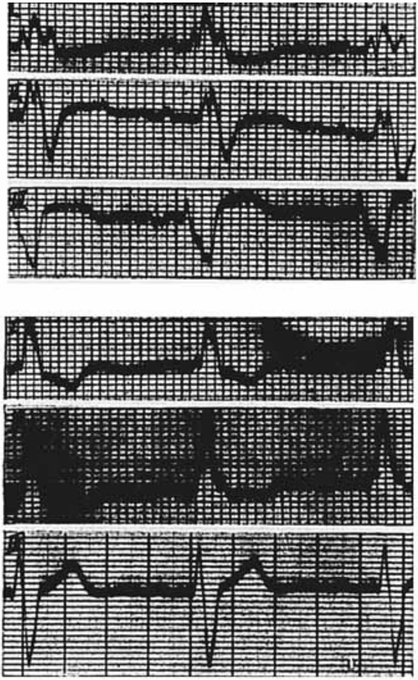

Fig. 3.—*Top:* Electrocardiogram taken April 14, 1920. *Bottom:* Electrocardiogram taken June 21, 1920.

The electrocardiogram taken at this time is shown in Fig. 5 (upper tracings). She returned to the clinic two weeks later. She was then markedly improved. The heart-rate was about 65 per minute and she could walk fairly comfortably. The electrocardiogram shown in Fig. 5, (lower) was taken at this time.

Three patients produced electrocardiograms in which the *PR* interval was slightly more than 0.2 second. Three had heart-block. In one of these the partial block of the main stem was transient (Fig. 6). In leads I and III there is seen a 2-1 auriculoventricular ratio. In lead II all the sinus impulses are passing over the bundle. The transmission period is, however, prolonged.

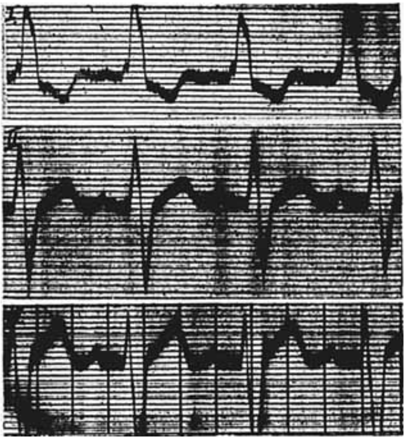

Fig. 4.—This record was made in October, 1920. The chief ventricular deflection is now tall and less atypical.

This patient had many attacks of Adams-Stokes syndrome and died suddenly from cardiac failure about six months following the time this electrocardiogram was taken.

In 5 instances there were auricular fibrillations.

Comment. All the patients of this series had symptoms and physical findings indicative of cardiac weakness. In 17 (48.5 per cent) these manifestations of weakness were very marked. That the outlook is poor is shown by the clinical course following the first examination. Eleven (52.3 per cent) of the 21 whose subsequent history was followed died within eighteen months, the average duration of life being less than one year. This mortality is in accord with the figures of other observers. Willius[3]

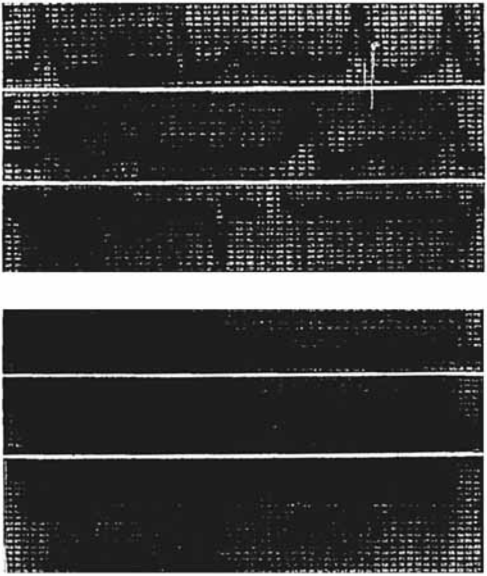

Fig. 5.—*Top:* Initial electrocardiogram; a few of the prevailing ventricular phases have an approximately normal duration; the others are very atypical and have a duration of about 0.15 second. *Bottom:* This electrocardiogram was taken two weeks later. The clinical condition of the patient was markedly improved.

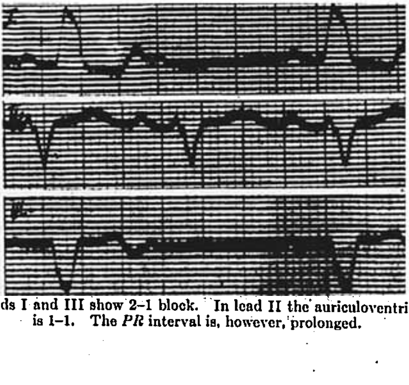

Fig. 6.—Leads I and III show 2–1 block. In lead II the auriculoventricular ratio is 1–1. The *PR* interval is, however, prolonged.

reported a mortality of 69.6 per cent in a series of 112 cases. The average duration of life in his cases from the time of examination was eight and one-half months. Forty-eight per cent of those studied by Oppenheimer and Rothschild[4] died within two years.

In those instances in which autopsies have been made there has usually been found an advanced sclerosis of the coronary arteries and extensive fibrous myocardial changes. The necropsy findings of 2 cases of this series, of 4 reported by Willius[3] and of 12 studied by Oppenheimer and Rothschild[4] were of this type. In 8 of the fourteen autopsies reported by Oppenheimer and Rothschild[4] there was found an occlusion of the anterior descending branch of the left coronary artery. Four of the cases of our series had severe attacks of anginal pain, suggestive of coronary artery accident, though there is no proof by autopsy of the correctness of such a diagnosis.[5] [6]

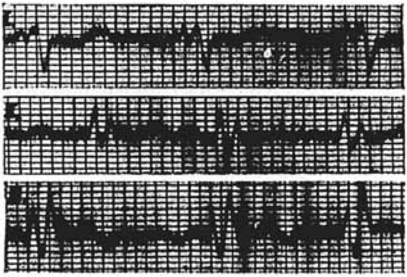

FIG. 7.—Shows very atypical *QRS* group. The duration is about 0.1 second.

It is to be noted from the histories, clinical findings and autopsy reports of these cases that the changes in the myocardium in a vast majority of the instances are chronic, degenerative in character and are often secondary to arteriosclerosis. The muscular changes are usually extensive. It is for this reason that these types of electrocardiograms are so rarely seen in cases of primary valvular disease. Only one instance of chronic valvular disease has been observed by us that gave an electrocardiogram (Fig. 7) similar to those under consideration. In this patient the myocardial invasion by the primary infection was quite extensive (autopsy).

Any operative procedure of a major character in a patient with this type of electrocardiogram should be considered dangerous even in the absence of advanced clinical cardiac manifestations. It will be noted that 3 of the patients of this group in whom the cardiac conditions were not considered alarming had major operations. Two had very difficult recoveries from cardiac upsets which developed rapidly after the operation. One died suddenly following the recovery from a prostatectomy, from what seemed to be a cardiac accident. Another died following operation for cataract from thrombosis of the cerebral arteries and of one femoral vein. Another died on the same day after the resection of the left fifth rib for an adhesive pericarditis.

The long duration of the *QRS* group and the direction assumed by the final deflection of these electrocardiograms probably indicate in most instances a block of the impulse over one branch or over a majority of the subdivisions of one branch of the auriculoventricular bundle. These conclusions are in harmony with experimental observations on the dog.[7 8 1 2] In the dog electrocardiograms characteristic of right or left branch block were produced only when these respective branches or a majority of the subdivisions of these branches were divided. The division of the arborization branches alone did not change the electrocardiogram.[7 8 1 2] There were some instances, however, following the cutting of the smaller ramifications to the left ventricle in which the electrocardiogram became atypical after the left ventricle was weakened and dilated as the result of the ligation of branches of the left coronary arteries. In some the electrocardiogram returned to normal after the ligatures were removed from the arteries.[2]

In 32 cases the electrocardiograms conform to the type usually considered as representing defects of the right division. The reasons for making a probable anatomic diagnosis on the basis of this type of electrocardiogram have been considered by one of us in a former report.[2] In this connection, however, it would seem that these points again deserve brief mention. The electrocardiograms under consideration resemble those produced experimentally in the dog by the division of the right branch of the auriculoventricular bundle. This is considered by Lewis[9] as the most convincing reason for making a probable anatomic diagnosis in man on the basis of the electrocardiographic findings. Furthermore, cases have been reported by Eppinger and Stoerk,[10] Cohn and Lewis[11] and Lewis[9] in which the suspected lesion of the right branch was found by histologic examination. Yet Cohn and Lewis[12] found no lesions of the right branch in four instances. In discussing these findings they suggested the possibility of a functional block [13 14 15 16] or in some instances of block of the main stem. Oppenheimer and Pardee[17] have recently recorded two cases in which the lesions were found in the branch other than that anticipated from the electrocardiograms. The findings in one of these cases are not sufficient to offset the above evidence. Further investigation along this line is desirable.

In one instance (Fig. 5, top) the duration of the *QRS* groups varied from 0.06 to 0.15 second. It is assumed that these findings represent a variation in the extent of the interventricular conduction defect. The possibility of incomplete bundle branch block was considered.

Three cases produced electrocardiograms in which the chief ventricular deflection was negative in lead I and positive in lead III. This type of electrocardiogram has been held to represent a defect of the left branch. This conception is not supported by experimental studies on the dog but is apparently based solely on a few clinical observations.

Summary. 1. Thirty-five cases were studied in which the electrocardiographic findings probably indicate a block of one main branch, or the majority of the subdivisions of one main branch, of the auriculoventricular bundle. The *QRS* groups were atypical in the three leads and exceeded 0.1 second in duration. In many the duration approached 0.15 second. The *T*-wave assumed the direction opposite to that of the prevailing ventricular deflection.

2. In 32 the prevailing ventricular deflection was positive in lead I and negative in lead II and the duration of the *QRS* was prolonged and in many instances approached 0.15 second duration. These electrocardiograms probably represent defects of the right branch.

3. In 3 cases there was an *S*-wave in lead I and *R*-wave in lead III. In these cases the location of the probable lesion of the conduction system is uncertain.

4. In some instances there was a change in the form of the *QRS* group which was coincident with the clinical improvement of the patients. In three there was an increase in the amplitude of the chief ventricular deflections. In one the atypical *QRS* groups disappeared except for a slight increase in duration of the *S*-wave in lead III.

5. All of these patients had symptoms of myocardial weakness. In 17, or 48.5 per cent, there were clinical manifestations of advanced cardiac disease. In nearly all instances the cardiac weakness seemed to be due to a slow degenerative change in the myocardium secondary to arteriosclerosis. Twelve, or 57.1 per cent, of the 21 patients that we were able to follow died within eighteen months. Ten of them died from cardiac failure. Autopsies were done in 3. In 2 there was an extensive sclerosis of the coronary arteries and marked myocardial fibrosis. In the other the anatomic diagnosis was obliterative pericarditis—huge dilatation and hypertrophy of the right auricle and ventricle, moderate hypertrophy of the left ventricle, myocardial fibrosis and cirrhosis of the liver.

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POST-TRAUMATIC CALCIFICATION OF THE PANCREAS, WITH DIABETES.

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The condition described in the above caption is, as far as some search through the literature seems to indicate, altogether unique, and hence a case illustrating it is herewith reported in some detail, especially since evidence bearing on the relation of the pancreas to diabetes is also of value.

The subject was a man, aged thirty-two years, a teamster, who while driving his wagon fell from the seat. As to whether the wheels of the wagon passed over his body or not the evidence of bystanders was conflicting, but from the nature of the injuries found at the autopsy there can be no doubt that they did. This happened on March 31, 1919, and he was at once taken to the Cook County Hospital. The examination on his admission showed him